

=> file hcaplus
COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
2.94	2.94

FULL ESTIMATED COST

FILE 'HCAPLUS' ENTERED AT 16:11:01 ON 10 DEC 2007
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FILE COVERS 1907 - 10 Dec 2007 VOL 147 ISS 25
FILE LAST UPDATED: 7 Dec 2007 (20071207/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s beta-glucan

1496636 BETA
15510 GLUCAN
L1 4854 BETA-GLUCAN
(BETA(W) GLUCAN)

=> s complement

L2 71149 COMPLEMENT

=> s antibody or monoclonal

321120 ANTIBODY
150577 MONOCLONAL
L3 363997 ANTIBODY OR MONOCLONAL

=> s barley

L4 52754 BARLEY

=> s l1 and l2

L5 146 L1 AND L2

=> s l1 and l2 and l4

L6 9 L1 AND L2 AND L4

=> s l1 and l3

L7 210 L1 AND L3

=> s l1 and l3 and l4

L8 17 L1 AND L3 AND L4

=> s l1 and l2 and l3

L9 48 L1 AND L2 AND L3

=> s l1 and l2 and l3 and l4

L10 4 L1 AND L2 AND L3 AND L4

=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.60	5.54

FILE 'STNGUIDE' ENTERED AT 16:11:12 ON 10 DEC 2007
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=> file hcaplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	5.60

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FILE COVERS 1907 - 10 Dec 2007 VOL 147 ISS 25
FILE LAST UPDATED: 7 Dec 2007 (20071207/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s l6 and (PY<2002 or AY<2002 or PRY<2002)

21937244 PY<2002
4193563 AY<2002
3670638 PRY<2002

L11 5 L6 AND (PY<2002 OR AY<2002 OR PRY<2002)

=> s l8 and (PY<2002 or AY<2002 or PRY<2002)

21937244 PY<2002
4193563 AY<2002
3670638 PRY<2002

L12 9 L8 AND (PY<2002 OR AY<2002 OR PRY<2002)

=> s l9 and (PY<2002 or AY<2002 or PRY<2002)

21937244 PY<2002
4193563 AY<2002
3670638 PRY<2002

L13 17 L9 AND (PY<2002 OR AY<2002 OR PRY<2002)

=> s l10 and (PY<2002 or AY<2002 or PRY<2002)

21937244 PY<2002
4193563 AY<2002
3670638 PRY<2002

L14 1 L10 AND (PY<2002 OR AY<2002 OR PRY<2002)

=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.60	8.20

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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Dec 7, 2007 (20071207/UP).

=> d l14 ti abs bib

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L14 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Specificity of membrane complement receptor type three (CR3) for
β-glucans
AB The binding of the iC3b receptor (CR3) to unopsonized zymosan resulted
from CR3 attachment to cell wall β-glucans. A specificity of
neutrophil responses for β -glucan was first
suggested by a comparison of yeast (*Saccharomyces cerevisiae*) cell wall
components for stimulation of a neutrophil superoxide burst. Three types
of expts. demonstrated a role for CR3 in these responses. First,
neutrophil ingestion of either yeast or yeast-derived β -
glucan particles was blocked by monoclonal anti-CR3,
fluid-phase iC3b, or soluble β -glucan from
barley. Monocyte ingestion of β -glucan
particles was also blocked by anti-CR3, but not by anti-CR1 or anti-C3.
Second, the neutrophil superoxide burst response to either zymosan or .
beta.-glucan particles was blocked by anti-CR3 or
fluid-phase iC3b, and was completely absent with neutrophils from 3
patients with an inherited deficiency of CR3. Third, CR3 was isolated
from solubilized neutrophils by affinity chromatog. on β -
glucan-Sepharose.
AN 1987:552442 HCAPLUS <<LOGINID::20071210>>
DN 107:152442
TI Specificity of membrane complement receptor type three (CR3) for
β-glucans
AU Ross, Gordon D.; Cain, Judith A.; Myones, Barry L.; Newman, Simon L.;
Lachmann, Peter J.
CS Dep. Med., Univ. North Carolina, Chapel Hill, NC, 27514, USA
SO Complement (1987), 4(2), 61-74
CODEN: CMPLDF; ISSN: 0253-5076
DT Journal

LA English

=> d l11 1-5 ti

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L11 ANSWER 1 OF 5 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Coniothyrium minitans β -(1,3) exoglucanase gene cbeG1

L11 ANSWER 2 OF 5 HCAPLUS COPYRIGHT 2007 ACS on STN
TI The β -glucan-binding lectin site of mouse CR3 (CD11b/CD18) and its function in generating a primed state of the receptor that mediates cytotoxic activation in response to iC3b-opsonized target cells

L11 ANSWER 3 OF 5 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Analysis of the sugar specificity and molecular location of the β -glucan-binding lectin site of complement receptor type 3 (CD11b/CD18)

L11 ANSWER 4 OF 5 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Specificity of membrane complement receptor type three (CR3) for β -glucans

L11 ANSWER 5 OF 5 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Activation of human polymorphonuclear leukocytes by particulate zymosan is related to both its major carbohydrate components: glucan and mannan

=> d l11 1-5 ti abs bib

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L11 ANSWER 1 OF 5 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Coniothyrium minitans β -(1,3) exoglucanase gene cbeG1
AB The invention provides the nucleotide sequence of a novel β -(1,3) exoglucanase gene denoted as cbeG1 of the soil-borne fungus Coniothyrium minitans. The deduced amino acid sequence of the encoded β -(1,3) exoglucanase enzyme, denoted CbeG1, is also provided. Encoded β -(1,3) exoglucanase CbeG1 is specific for the substrate laminarin, in that results showed no activity with other substrates tested, such as CM-cellulose, barley β -glucan, lichenan, oat spelt xylan and birchwood xylan. The pH and temperature optima for β -(1,3) exoglucanase CbeG1 are 6.0 and 57° C., resp. CbeG1 contains 784 amino acids, and has a predicted isoelec. point (pI) of 6.0 and mol. weight of 83,646 Daltons. The invention further provides vectors and cells comprising a nucleic acid mol. encoding the cbeG1 gene, and methods for producing β -(1,3) exoglucanase CbeG1. The cbeG1 gene is compatible with a eukaryotic heterologous expression system, making it particularly useful for a wide range of industrial applications, such as improvement of plant resistance to fungal phytopathogens or use in ruminant microbial transgenic strategies to improve feed digestion and nutritive carbohydrate availability from forage feed. In addition, the high activity of CbeG1 over broad pH and temperature ranges may be beneficial for use

in high temperature industrial applications, such as bleaching of pulp, which require temps. greater than 37° C. Further, CbeG1 may complement degradation initiated by endoglucanases which release oligoglucans, in that β -(1,3) exoglucanase sequentially hydrolyzes β -(1,3) glucan fragments and is required to hydrolyze oligoglucan fragments completely to obtain D-glucose, which can be assimilated.

AN 2003:473330 HCAPLUS <<LOGINID::20071210>>
 DN 139:48173
 TI Coniothyrium minitans β -(1,3) exoglucanase gene cbeG1
 IN Laroche, Andre J.; Huang, Timothy Yikai; Frick, Michele M.; Lu, Zhen-Xiang; Huang, Hung Chang; Cheng, Kuo Joan
 PA Her Majesty the Queen in Right of Canada, as Represented by the Minister of Agriculture and Agrifood, Can.
 SO U.S. Pat. Appl. Publ., 43 pp.
 CODEN: USXXCO
 DT Patent
 LA English
 FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2003115627	A1	20030619	US 2000-733643	20001208 <--
	US 6734344	B2	20040511		
	CA 2325774	A1	20010610	CA 2000-2325774	20001208 <--
PRAI	US 1999-170168P	P	19991210	<--	

RE.CNT 71 THERE ARE 71 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 2 OF 5 HCAPLUS COPYRIGHT 2007 ACS on STN
 TI The β -glucan-binding lectin site of mouse CR3 (CD11b/CD18) and its function in generating a primed state of the receptor that mediates cytotoxic activation in response to iC3b-opsonized target cells
 AB Mouse leukocyte CR3 (Mac-1, α M β 2 integrin) was shown to function as a receptor for β -glucans in the same way as human CR3. Soluble zymosan polysaccharide (SZP) or pure β -glucans labeled with FITC or 125I bound in a saturable and reversible manner to neutrophils, macrophages, and NK cells. This lectin activity was blocked by anti-CD11b mAb M1/70 or 5C6 and did not occur with leukocytes from CR3-/- (CD11b-deficient) mice. SZP preps. containing primarily mannose or glucose bound to CR3, and the binding of 125I-labeled β -glucan to CR3 was competitively inhibited by β -glucans from barley or seaweed, but not by yeast α -mannan. Also, as with human CR3, the lectin site of mouse CR3 was inhibited by α - or β -methylglucoside (but not D-glucose), α - or β -methylmannoside, and N-acetyl-D-glucosamine. Phagocytosis of zymosan and serum-opsonized zymosan was partially inhibited by anti-CR3 and was reduced to <40% of normal with leukocytes from CR3-/- mice. As with neutrophils from patients with CD18 deficiency, neutrophils from CR3-/- mice exhibited no phagocytosis of particulate β -glucan. SZP or β -glucans primed CR3 of neutrophils, macrophages, and NK cells for cytotoxicity of iC3b-opsonized tumor cells that otherwise did not trigger killing. β -Glucan priming for cytotoxicity was inhibited by anti-CR3 and did not occur with leukocytes from CR3-/- mice. The primed state of macrophage and NK cell CR3 remained detectable for 18 to 24 h after pulsing with β -glucans. The similarity of mouse and human CR3 in response to β -glucans highlights the utility of mouse tumor models for development of therapeutic β -glucans.

AN 1999:107663 HCAPLUS <<LOGINID::20071210>>
 DN 130:280682
 TI The β -glucan-binding lectin site of mouse CR3 (CD11b/CD18) and its function in generating a primed state of the receptor that mediates cytotoxic activation in response to iC3b-opsonized target cells
 AU Xia, Yu; Vetvicka, Vclav; Yan, Jun; Hanikyrova, Margareta; Mayadas, Tanya; Ross, Gordon D.
 CS Division of Experimental Immunology and Immunopathology, Department of Pathology, and Department of Microbiology and Immunology, University of Louisville, Louisville, KY, 40292, USA
 SO Journal of Immunology (1999), 162(4), 2281-2290

CODEN: JOIMA3; ISSN: 0022-1767

PB American Association of Immunologists

DT Journal

LA English

RE.CNT 83 THERE ARE 83 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 3 OF 5 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Analysis of the sugar specificity and molecular location of the .
beta.-glucan-binding lectin site of complement
receptor type 3 (CD11b/CD18)

AB Zymosan, the cell wall from *Saccharomyces cerevisiae*, was reported to be a
macrophage activator through its β -glycan over 30 yr ago.
Nevertheless, the identity of the β -glucan
receptor has been controversial. This study showed that the
 α M β 2-integrin, CR3 (Mac-1, CD11b/CD18) served as the .
beta.-glucan receptor through one or more lectin sites
located outside of the CD11b I-domain that contains the binding sites for
iC3b, ICAM-1, and fibrinogen. Sugar specificity, analyzed with
FITC-labeled soluble polysaccharides and flow cytometry, showed CR3-specific
staining with several pure β -glucans but not with α -mannan.
However, a 10-kDa soluble zymosan polysaccharide (SZP) with high affinity
(6.7×10^{-8} M) for CR3 consisted largely of mannose and .apprx.5%
glucose. Binding of either SZP-FITC or β -glucan
-FITC to CR3 was blocked not only by pure β -glucans from yeast,
mushroom, seaweed, or barley, but also by N-acetyl-D-glucosamine
(NADG), α - or β -methylmannoside, and α - or
 β -methylglucoside. SZP-FITC and β -glucan
-FITC stained all leukocyte types similarly to anti-CR3-FITC, and
polysaccharide-FITC staining was inhibited $\geq 95\%$ by unlabeled
anti-CR3. SZP-FITC staining of cells expressing recombinant chimeras
between CR3 and CR4 (p150,95, CD11c/CD18) suggested that both the divalent
cation-binding region of CD11b and the region C-terminal to it may
regulate binding of polysaccharides to CR3. Unlabeled SZP or .
beta.-glucan also blocked CR3 staining by 11 mAb to
C-terminal domain epitopes of CD11b but had no effect on staining by mAb
directed to the I-domain. In conclusion, CR3 serves as the leukocyte .
beta.-glucan receptor through a cation-independent
lectin site located C-terminal to the I-domain of CD11b. Its sugar
specificity is broader than originally appreciated, allowing it to react
with certain polysaccharides containing mannose or NADG, as well as glucose.

AN 1996:63811 HCAPLUS <<LOGINID::20071210>>

DN 124:114996

TI Analysis of the sugar specificity and molecular location of the .
beta.-glucan-binding lectin site of complement
receptor type 3 (CD11b/CD18)

AU Thornton, Brian P.; Vetvicka, Vaclav; Pitman, Mark; Goldman, Robert C.;
Ross, Gordon D.

CS Dep. Pathol., Univ. Louisville, Louisville, KY, 40292, USA

SO Journal of Immunology (1996), 156(3), 1235-46

CODEN: JOIMA3; ISSN: 0022-1767

PB American Association of Immunologists

DT Journal

LA English

L11 ANSWER 4 OF 5 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Specificity of membrane complement receptor type three (CR3) for
 β -glucans

AB The binding of the iC3b receptor (CR3) to unopsonized zymosan resulted
from CR3 attachment to cell wall β -glucans. A specificity of
neutrophil responses for β -glucan was first
suggested by a comparison of yeast (*Saccharomyces cerevisiae*) cell wall
components for stimulation of a neutrophil superoxide burst. Three types
of expts. demonstrated a role for CR3 in these responses. First,

neutrophil ingestion of either yeast or yeast-derived β -glucan particles was blocked by monoclonal anti-CR3, fluid-phase iC3b, or soluble β -glucan from barley. Monocyte ingestion of β -glucan particles was also blocked by anti-CR3, but not by anti-CR1 or anti-C3. Second, the neutrophil superoxide burst response to either zymosan or β -glucan particles was blocked by anti-CR3 or fluid-phase iC3b, and was completely absent with neutrophils from 3 patients with an inherited deficiency of CR3. Third, CR3 was isolated from solubilized neutrophils by affinity chromatog. on β -glucan -Sephrose.

AN 1987:552442 HCAPLUS <<LOGINID::20071210>>
DN 107:152442
TI Specificity of membrane complement receptor type three (CR3) for β -glucans
AU Ross, Gordon D.; Cain, Judith A.; Myones, Barry L.; Newman, Simon L.; Lachmann, Peter J.
CS Dep. Med., Univ. North Carolina, Chapel Hill, NC, 27514, USA
SO Complement (1987), 4(2), 61-74
CODEN: CMPLDF; ISSN: 0253-5076
DT Journal
LA English

L11 ANSWER 5 OF 5 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Activation of human polymorphonuclear leukocytes by particulate zymosan is related to both its major carbohydrate components: glucan and mannan
AB Unopsonized particulate zymosan and its major carbohydrate component glucan were phagocytosed under serum-free conditions by adherent polymorphonuclear leukocytes (PMN) in a dose- and time-dependent manner. Preincubation of PMN monolayers with mannan did not cause a reduction in the phagocytosis of either particle. The phagocytic response was inhibited by preincubation of the cells with trypsin at a concentration that did not inhibit the phagocytosis of sheep erythrocytes coated with IgG or of latex particles. Homol. of the recognition mechanisms for glucan and zymosan was confirmed when cells cultured on fixed glucan or on fixed zymosan failed to ingest either particle to more than 40% of control phagocytosis. Similarly, zymosan and glucan activated PMN in suspension, in a dose- and time-dependent manner, to generate reactive O species which were measured as luminol-dependent chemiluminescence (CL). There was however, a 4-fold greater CL response to zymosan. Preincubation of PMN with mannan resulted in a decreased CL response to zymosan, while the response to glucan was unaffected. The CL response was also sensitive to a range of concns. of trypsin. In contrast, 2 other complex polysaccharide particles (barley-derived β -glucan and algae-derived laminarin) were not phagocytosed by PMN, nor did they cause the generation of CL, despite the fact that they possessed the capacity, in common with zymosan and glucan, to activate the alternative pathway of complement. The identification of a trypsin-sensitive recognition mechanism on the surface of human PMN for unopsonized zymosan and glucan represents a response not hitherto characterized. Furthermore, the phagocytosis of unopsonized zymosan by human PMN is dependent primarily on its glucan content, but its capacity to activate the respiratory burst may involve mannan and the recruitment of a second cell surface recognition mechanism.

AN 1986:404970 HCAPLUS <<LOGINID::20071210>>
DN 105:4970
TI Activation of human polymorphonuclear leukocytes by particulate zymosan is related to both its major carbohydrate components: glucan and mannan
AU Williams, J. D.; Topley, N.; Alobaidi, H. M.; Harber, M. J.
CS KRUF Inst., R. Infirm., Cardiff, UK
SO Immunology (1986), 58(1), 117-24
CODEN: IMMUAM; ISSN: 0019-2805
DT Journal
LA English

=> d l12 1-9 ti

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L12 ANSWER 1 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN

TI The location of (1-3)- β -glucan in the nucellar projection and in the vascular tissue of the crease in developing barley grain using a (1-3)- β -glucan-specific monoclonal antibody

L12 ANSWER 2 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Immunocytochemical location of the (1 \rightarrow 3) (1 \rightarrow 4)-.beta.-glucan lichenin in the lichen-forming ascomycete *Cetraria islandica* (Icelandic moss)

L12 ANSWER 3 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Purification and characterization of a (1 \rightarrow 3)- β -D-glucan-binding protein from horseshoe crab (*Tachypleus tridentatus*) amoebocytes

L12 ANSWER 4 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Purification and characterization of (1 \rightarrow 3, 1 \rightarrow 4)-.beta.-glucan endohydrolases from germinated wheat (*Triticum aestivum*)

L12 ANSWER 5 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Development and regulation of (1 \rightarrow 3,1 \rightarrow 4)- β -glucan endohydrolases in germinating wheat (*Triticum aestivum*)

L12 ANSWER 6 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Identification of individual (1 \rightarrow 3,1 \rightarrow 4)- β -D-glucanase isoenzymes in extracts of germinated barley using specific monoclonal antibodies

L12 ANSWER 7 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Specificity of membrane complement receptor type three (CR3) for β -glucans

L12 ANSWER 8 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Visualization of α -amylase movement and cell wall breakdown during barley malting - practical application of current research

L12 ANSWER 9 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Cross reactions of polyglucoses in antipneumococcal serums. VI. Precipitation of type VIII and type III antiserums by β -glucans

=> d l12 d his

'D' IS NOT VALID HERE

For an explanation, enter "HELP DISPLAY HISTORY".

=> d his

(FILE 'HOME' ENTERED AT 16:02:28 ON 10 DEC 2007)

FILE 'HCAPLUS' ENTERED AT 16:11:01 ON 10 DEC 2007

L1 4854 S BETA-GLUCAN
L2 71149 S COMPLEMENT
L3 363997 S ANTIBODY OR MONOCLONAL
L4 52754 S BARLEY
L5 146 S L1 AND L2
L6 9 S L1 AND L2 AND L4

L7 210 S L1 AND L3
L8 17 S L1 AND L3 AND L4
L9 48 S L1 AND L2 AND L3
L10 4 S L1 AND L2 AND L3 AND L4

FILE 'STNGUIDE' ENTERED AT 16:11:12 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 16:11:58 ON 10 DEC 2007

L11 5 S L6 AND (PY<2002 OR AY<2002 OR PRY<2002)
L12 9 S L8 AND (PY<2002 OR AY<2002 OR PRY<2002)
L13 17 S L9 AND (PY<2002 OR AY<2002 OR PRY<2002)
L14 1 S L10 AND (PY<2002 OR AY<2002 OR PRY<2002)

FILE 'STNGUIDE' ENTERED AT 16:12:11 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 16:12:19 ON 10 DEC 2007

FILE 'STNGUIDE' ENTERED AT 16:12:20 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 16:12:51 ON 10 DEC 2007

FILE 'STNGUIDE' ENTERED AT 16:12:52 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 16:13:10 ON 10 DEC 2007

FILE 'STNGUIDE' ENTERED AT 16:13:10 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 16:13:26 ON 10 DEC 2007

FILE 'STNGUIDE' ENTERED AT 16:13:26 ON 10 DEC 2007

=> file hcaplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.24	40.82
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-4.68

FILE 'HCAPLUS' ENTERED AT 16:16:02 ON 10 DEC 2007

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FILE COVERS 1907 - 10 Dec 2007 VOL 147 ISS 25

FILE LAST UPDATED: 7 Dec 2007 (20071207/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s (cancer or tumor or neoplas?)

339575 CANCER
432104 TUMOR
520672 NEOPLAS?

L15 795839 (CANCER OR TUMOR OR NEOPLAS?)

=> s l15 and l5

L16 57 L15 AND L5

=> s l15 and l11

L17 1 L15 AND L11

=> s l15 and l7

L18 57 L15 AND L7

=> s l15 and l12

L19 0 L15 AND L12

=> s l15 and l13

L20 6 L15 AND L13

=> s l15 and l14

L21 0 L15 AND L14

=> s l16 and (PY<2002 or AY<2002 or PRY<2002)

21937244 PY<2002
4193563 AY<2002
3670638 PRY<2002

L22 29 L16 AND (PY<2002 OR AY<2002 OR PRY<2002)

=> s l17 and (PY<2002 or AY<2002 or PRY<2002)

21937244 PY<2002
4193563 AY<2002
3670638 PRY<2002

L23 1 L17 AND (PY<2002 OR AY<2002 OR PRY<2002)

=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	2.60	43.42
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
	ENTRY	SESSION
CA SUBSCRIBER PRICE	0.00	-4.68

FILE 'STNGUIDE' ENTERED AT 16:16:17 ON 10 DEC 2007
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LAST RELOADED: Dec 7, 2007 (20071207/UP).

=> d l17 ti

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L17 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2007 ACS on STN

TI The β -glucan-binding lectin site of mouse CR3 (CD11b/CD18) and its function in generating a primed state of the receptor that mediates cytotoxic activation in response to iC3b-opsonized target cells

=> d l17 ti abs bib

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L17 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2007 ACS on STN

TI The β -glucan-binding lectin site of mouse CR3 (CD11b/CD18) and its function in generating a primed state of the receptor that mediates cytotoxic activation in response to iC3b-opsonized target cells

AB Mouse leukocyte CR3 (Mac-1, α M β 2 integrin) was shown to function as a receptor for β -glucans in the same way as human CR3. Soluble zymosan polysaccharide (SZP) or pure β -glucans labeled with FITC or 125I bound in a saturable and reversible manner to neutrophils, macrophages, and NK cells. This lectin activity was blocked by anti-CD11b mAb M1/70 or 5C6 and did not occur with leukocytes from CR3-/- (CD11b-deficient) mice. SZP preps. containing primarily mannose or glucose bound to CR3, and the binding of 125I-labeled β -glucan to CR3 was competitively inhibited by β -glucans from barley or seaweed, but not by yeast α -mannan. Also, as with human CR3, the lectin site of mouse CR3 was inhibited by α - or β -methylglucoside (but not D-glucose), α - or β -methylmannoside, and N-acetyl-D-glucosamine. Phagocytosis of zymosan and serum-opsonized zymosan was partially inhibited by anti-CR3 and was reduced to <40% of normal with leukocytes from CR3-/- mice. As with neutrophils from patients with CD18 deficiency, neutrophils from CR3-/- mice exhibited no phagocytosis of particulate β -glucan. SZP or β -glucans primed CR3 of neutrophils, macrophages, and NK cells for cytotoxicity of iC3b-opsonized tumor cells that otherwise did not trigger killing. β -Glucan priming for cytotoxicity was inhibited by anti-CR3 and did not occur with leukocytes from CR3-/- mice. The primed state of macrophage and NK cell CR3 remained detectable for 18 to 24 h after pulsing with β -glucans. The similarity of mouse and human CR3 in response to β -glucans highlights the utility of mouse tumor models for development of therapeutic β -glucans.

AN 1999:107663 HCAPLUS <<LOGINID::20071210>>

DN 130:280682

TI The β -glucan-binding lectin site of mouse CR3 (CD11b/CD18) and its function in generating a primed state of the receptor that mediates cytotoxic activation in response to iC3b-opsonized target cells

AU Xia, Yu; Vetvicka, Vclav; Yan, Jun; Hanikyrova, Margareta; Mayadas, Tanya; Ross, Gordon D.

CS Division of Experimental Immunology and Immunopathology, Department of Pathology, and Department of Microbiology and Immunology, University of Louisville, Louisville, KY, 40292, USA

SO Journal of Immunology (1999), 162(4), 2281-2290
CODEN: JOIMA3; ISSN: 0022-1767

PB American Association of Immunologists

DT Journal

LA English

RE.CNT 83 THERE ARE 83 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d 120 1-6 ti abs bib

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L20 ANSWER 1 OF 6 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Antitumor antibody-enhancing glucan

AB This invention provides a composition comprising an effective amount of glucan capable of enhancing efficacy of antibodies. This invention further provides the above compns. and a pharmaceutically acceptable carrier. This invention also provides a method for treating a subject with cancer comprising administering the above-described composition comprising effective amount of glucan capable of enhancing efficacy of vaccines. This invention provides a composition comprising effective amount of glucan capable of enhancing efficacy of vaccines. This invention also provides a method of treating a subject comprising administering the above pharmaceutical composition to the subject. This invention provides a composition comprising effective amount of glucan capable of enhancing efficacy of natural antibodies. This invention provides a composition comprising effective amount of glucan capable of enhancing host immunity. This invention also provides a composition comprising effective amount of glucan capable of enhancing the action of an agent in preventing tissue rejection. It was shown that β -glucans greatly enhanced the antitumor effects of monoclonal antibodies against established tumors in mice.

AN 2002:574940 HCAPLUS <<LOGINID::20071210>>

DN 137:119657

TI Antitumor antibody-enhancing glucan

IN Cheung, Nai-Kong V.

PA Sloan-Kettering Institute for Cancer Research, USA

SO PCT Int. Appl., 114 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 3

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PI	WO 2002058711	A1	20020801	WO 2002-US1276	20020115 <--
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	CA 2434938	A1	20020801	CA 2002-2434938	20020115 <--
	AU 2002241905	A1	20020806	AU 2002-241905	20020115 <--
	EP 1357919	A1	20031105	EP 2002-707502	20020115 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	US 2004116379	A1	20040617	US 2003-621027	20030716 <--
	US 2006020128	A1	20060126	US 2005-218044	20050831 <--
	US 2006160766	A1	20060720	US 2006-334763	20060117 <--
PRAI	US 2001-261911P	P	20010116	<--	
	WO 2002-US1276	W	20020115		
	US 2003-621027	A1	20030716		
	WO 2004-US23099	A2	20040716		
	US 2005-218044	A2	20050831		

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 2 OF 6 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Clostridial neurotoxin targeted conjugates for inhibition of secretion from non-neuronal cells

AB A method of treatment of disease by inhibition of cellular secretory processes is provided. The method has particular application in the treatment of diseases dependent on the exocytotic activity of endocrine cells, exocrine cells, inflammatory cells, cells of the immune system, cells of the cardiovascular system, and bone cells. Agents and compns. therefor, as well as methods for manufacturing these agents and compns., are provided. In a preferred embodiment a clostridial neurotoxin, substantially devoid of holotoxin binding affinity for neuronal cells of the presynaptic muscular junction, is associated with a targeting moiety. The targeting moiety is selected such that the clostridial toxin conjugate so formed may be directed to a non-neuronal target cell to which the conjugate may bind. Following binding, a neurotoxin component of the conjugate, which is capable of inhibition of cellular secretion, passes into the cytosol of the target cell by cellular internalization mechanisms. Thereafter, inhibition of secretion from the target cell is effected.

AN 2001:228744 HCAPLUS <<LOGINID::20071210>>

DN 134:247267

TI Clostridial neurotoxin targeted conjugates for inhibition of secretion from non-neuronal cells

IN Foster, Keith Alan; Chaddock, John Andrew; Purkiss, John Robert; Quinn, Conrad Padraig

PA Microbiological Research Authority, UK

SO PCT Int. Appl., 63 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001021213	A2	20010329	WO 2000-GB3669	20000925 <--
	WO 2001021213	A3	20020711		
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	RW:			GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG	
	CA 2383470	A1	20010329	CA 2000-2383470	20000925 <--
EP	1235594	A2	20020904	EP 2000-962721	20000925 <--
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	JP 2003509476	T	20030311	JP 2001-524636	20000925 <--
	AU 782457	B2	20050728	AU 2000-74365	20000925 <--
	US 2003180289	A1	20030925	US 2002-88665	20020814 <--
	AU 2005227383	A1	20051124	AU 2005-227383	20051027 <--
	US 2006216283	A1	20060928	US 2006-327855	20060109 <--
PRAI	GB 1999-22554	A	19990923	<--	
	WO 2000-GB3669	W	20000925	<--	
	WO 2000-GB3681	A	20000925	<--	
	US 2002-88665	A1	20020814		

L20 ANSWER 3 OF 6 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Immunopharmacological and immunotoxicological activities of a water-soluble (1 → 3)-β-D-glucan, CSBG from Candida spp

AB We have established a convenient, two-step procedure to solubilize the yeast cell wall (1→3)-β-D-glucan using the combination of

NaClO oxidation and DMSO extraction Candida soluble β -D-glucan (CSBG) was mainly composed of a linear β -1,3 glucan with a linear β -1,6-glucan moiety. In this study, we screened for several immunopharmacol. activities of CSBG and found the following activities: (1) interleukin-6 synthesis of macrophages in vitro; (2) antagonistic effect for zymosan mediated-tumor necrosis factor synthesis of macrophages; (3) augmentation for lipopolysaccharide mediated tumor necrosis factor and nitrogen oxide syntheses of macrophages; (4) activation of alternative pathway of complement; (5) hematopoietic response on cyclophosphamide induced leukopenia; (6) the antitumor effect on ascites form tumor; (7) Enhanced vascular permeability; (8) priming effect on lipopolysaccharide triggered TNF- α synthesis; and (9) adjuvant effect on antibody production. These results strongly suggested that CSBG possessed various immunopharmacol. activity.

AN 2000:235041 HCAPLUS <<LOGINID::20071210>>

DN 133:12504

TI Immunopharmacological and immunotoxicological activities of a water-soluble (1 \rightarrow 3)- β -D-glucan, CSBG from Candida spp

AU Tokunaka, Kazuhiro; Ohno, Naohito; Adachi, Yoshiyuki; Tanaka, Shigenori; Tamura, Hiroshi; Yadomae, Toshiro

CS Laboratory for Immunopharmacology of Microbial Products, School of Pharmacy, Tokyo University of Pharmacy and Life Science, Tokyo, 192-0392, Japan

SO International Journal of Immunopharmacology (2000), 22(5), 383-394

CODEN: IJIMDS; ISSN: 0192-0561

PB Elsevier Science Ltd.

DT Journal

LA English

RE.CNT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 4 OF 6 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Interactions of Penicillium marneffei with human leukocytes in vitro

AB Penicillium marneffei, a dimorphic fungus endemic in parts of Asia, causes disease in those with impaired cell-mediated immunity, especially persons with AIDS. The histopathol. of penicilliosis marneffei features the intracellular infection of macrophages. The authors studied the interactions between human leukocytes and heat-killed yeast-phase P. marneffei. Monocyte-derived macrophages bound and internalized P. marneffei in the presence of complement-sufficient pooled human serum (PHS). Binding and phagocytosis were still seen if PHS was heat inactivated or omitted altogether. The binding of unopsonized P. marneffei to monocyte-derived macrophages occurred in the absence of divalent cations and was not affected by inhibitors of mannose and β -glucan receptors or monoclonal antibodies directed against CD14 and CD11/CD18. Binding was profoundly inhibited by wheat germ agglutinin. A vigorous respiratory burst was seen in peripheral blood mononuclear cells (PBMC) stimulated with P. marneffei, regardless of whether the fungi were opsonized. However, tumor necrosis factor alpha (TNF- α) release from PBMC stimulated with P. marneffei occurred only if serum was present. These data demonstrate that (i) monocyte-derived macrophages bind and phagocytose P. marneffei even in the absence of opsonization, (ii) binding is divalent cation independent but is inhibited by wheat germ agglutinin, suggesting that the major receptor(s) recognizing P. marneffei is a glycoprotein with exposed N-acetyl- β -D-glucosaminyl groups, (iii) P. marneffei stimulates the respiratory burst regardless of whether opsonins are present, and (iv) serum factors are required for P. marneffei to stimulate TNF- α release. The ability of unopsonized P. marneffei to parasitize mononuclear phagocytes without stimulating the production of TNF- α may be critical for the virulence of this intracellular parasite.

AN 1999:554591 HCAPLUS <<LOGINID::20071210>>

DN 131:285214
TI Interactions of *Penicillium marneffei* with human leukocytes in vitro
AU Rongrungruang, Yong; Levitz, Stuart M.
CS The Evans Memorial Department of Clinical Research and the Department of
Medicine, Boston University School of Medicine, Boston, MA, 02118, USA
SO Infection and Immunity (1999), 67(9), 4732-4736
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
RE.CNT 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 5 OF 6 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Effect of lentinan and mannan on phagocytosis of fluorescent latex
microbeads by mouse peritoneal macrophages: a flow cytometric study
AB Lentinan, an immunopotentiating β -1,3-glucan polysaccharide stimulated
the in vitro phagocytosis of BSA-coated, C3b- or monoclonal
immunoglobulin (IgG2b)-coated fluorescent microspheres by resident or
thioglycollate-elicited mouse macrophages in the dose-dependent manner.
Anal. of flow cytometric data has shown that microbead phagocytosis of
resident macrophages, which exhibit a lower basic phagocytic activity than
the thioglycollate elicited ones, has been augmented by up to 900% due to
lentinan. The percent ratio of phagocytes among peritoneal exudate cells,
however, remained unchanged after short-term lentinan stimulation.
Preincubation of the cells with lentinan resulted in increased ingestion
of the microbeads. Activation of phagocytosis by lentinan is therefore
due in part to the direct stimulation of the cells, however, lentinan also
serves as supplementary opsonin for complement C3b-coated beads.
Mannan inhibited the ingestion of C3b-coated microspheres by 75%, which
was abolished in part when lentinan was also added to the cells. Mannan
did not influence the phagocytosis of BSA-coated or IgG-coated beads.
These data, based solely on in vitro studies, suggest a β -
glucan receptor mediated activation of phagocytes by lentinan.
These receptors are different from the C3b, Fc or mannose receptors. It
is very likely that stimulation of phagocytic activity of macrophages by
lentinan may contribute to the antitumor action of this immunopotentiating
polysaccharide.

AN 1989:630536 HCAPLUS <<LOGINID::20071210>>
DN 111:230536
TI Effect of lentinan and mannan on phagocytosis of fluorescent latex
microbeads by mouse peritoneal macrophages: a flow cytometric study
AU Abel, Gyorgy; Szollosi, Janos; Chihara, Goro; Fachet, Jozsef
CS Inst. Pathophysiol., Univ. Med. Sch., Debrecen, Hung.
SO International Journal of Immunopharmacology (1989), 11(6),
615-21
CODEN: IJIMDS; ISSN: 0192-0561
DT Journal
LA English

L20 ANSWER 6 OF 6 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Antitumor and immunomodulating activities of a β -
glucan obtained from liquid-cultured *Grifola frondosa*
AB The effects of the β -1,3-glucan, LELFD, obtained from liquid-cultured
mycelium of *G. frondosa*, on the growth of syngeneic tumors and immune
responses in mice were examined. In Meth A fibrosarcoma or IMC carcinoma
solid tumor systems, LELFD administered i.p. or intralesionally
(i.l.) exhibited significant antitumor effects. However, the growth of
L1210 and P388 leukemias was unaffected by the injection of LELFD. The
injection of LELFD i.p. enhanced the activities of natural killer cells
and macrophages in mice. LELFD also enhanced the antibody
response when it was injected i.p. with sheep red blood cells into mice.
Furthermore, it was found that LELFD could activate complement
pathway.

AN 1989:185485 HCAPLUS <<LOGINID::20071210>>
DN 110:185485
TI Antitumor and immunomodulating activities of a β -
glucan obtained from liquid-cultured Grifola frondosa
AU Suzuki, Iwao; Hashimoto, Koichi; Oikawa, Shozo; Sato, Kichiro; Osawa,
Masumi; Yadomae, Toshiro
CS Tokyo Coll. Pharm.; Hachioji, 192-03, Japan
SO Chemical & Pharmaceutical Bulletin (1989), 37(2), 410-13
CODEN: CPBTAL; ISSN: 0009-2363
DT Journal
LA English

=> d his

(FILE 'HOME' ENTERED AT 16:02:28 ON 10 DEC 2007)

FILE 'HCAPLUS' ENTERED AT 16:11:01 ON 10 DEC 2007

L1 4854 S BETA-GLUCAN
L2 71149 S COMPLEMENT
L3 363997 S ANTIBODY OR MONOCLONAL
L4 52754 S BARLEY
L5 146 S L1 AND L2
L6 9 S L1 AND L2 AND L4
L7 210 S L1 AND L3
L8 17 S L1 AND L3 AND L4
L9 48 S L1 AND L2 AND L3
L10 4 S L1 AND L2 AND L3 AND L4

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L11 5 S L6 AND (PY<2002 OR AY<2002 OR PRY<2002)
L12 9 S L8 AND (PY<2002 OR AY<2002 OR PRY<2002)
L13 17 S L9 AND (PY<2002 OR AY<2002 OR PRY<2002)
L14 1 S L10 AND (PY<2002 OR AY<2002 OR PRY<2002)

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FILE 'STNGUIDE' ENTERED AT 16:12:52 ON 10 DEC 2007

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FILE 'STNGUIDE' ENTERED AT 16:13:10 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 16:13:26 ON 10 DEC 2007

FILE 'STNGUIDE' ENTERED AT 16:13:26 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 16:16:02 ON 10 DEC 2007

L15 795839 S (CANCER OR TUMOR OR NEOPLAS?)
L16 57 S L15 AND L5
L17 1 S L15 AND L11
L18 57 S L15 AND L7
L19 0 S L15 AND L12
L20 6 S L15 AND L13
L21 0 S L15 AND L14
L22 29 S L16 AND (PY<2002 OR AY<2002 OR PRY<2002)

L23 1 S L17 AND (PY<2002 OR AY<2002 OR PRY<2002)

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For an explanation, enter "HELP LOGOFF".

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CA SUBSCRIBER PRICE	0.00	-10.14

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Connecting via Winsock to STN

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PASSWORD:
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FILE 'STNGUIDE' ENTERED AT 17:05:36 ON 10 DEC 2007
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FULL ESTIMATED COST	0.06	71.79
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CA SUBSCRIBER PRICE	0.00	-10.14

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CA SUBSCRIBER PRICE	0.00	-10.14

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 FILE LAST UPDATED: 7 Dec 2007 (20071207/ED)

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This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s barley or oat or wheat or moss

52754 BARLEY

19192 OAT

133570 WHEAT

7925 MOSS

L24 191167 BARLEY OR OAT OR WHEAT OR MOSS

=> s l24 and l1 and l2

L25 11 L24 AND L1 AND L2

=> s l24 and l1 and l2 and l15

L26 5 L24 AND L1 AND L2 AND L15

=> s l24 and l1 and l3

L27 25 L24 AND L1 AND L3

=> s l24 and l1 and l3 and l15

L28 5 L24 AND L1 AND L3 AND L15

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21937244 PY<2002

4193563 AY<2002

3670638 PRY<2002

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21937244 PY<2002

4193563 AY<2002

3670638 PRY<2002

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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Dec 7, 2007 (20071207/UP).

=> d l30 1-3 ti abs bib

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L30 ANSWER 1 OF 3 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Clostridial neurotoxin targeted conjugates for inhibition of secretion
from non-neuronal cells
AB A method of treatment of disease by inhibition of cellular secretory
processes is provided. The method has particular application in the
treatment of diseases dependent on the exocytotic activity of endocrine
cells, exocrine cells, inflammatory cells, cells of the immune system,
cells of the cardiovascular system, and bone cells. Agents and compns.
therefor, as well as methods for manufacturing these agents and compns., are
provided. In a preferred embodiment a clostridial neurotoxin,
substantially devoid of holotoxin binding affinity for neuronal cells of
the presynaptic muscular junction, is associated with a targeting moiety.
The targeting moiety is selected such that the clostridial toxin conjugate
so formed may be directed to a non-neuronal target cell to which the
conjugate may bind. Following binding, a neurotoxin component of the
conjugate, which is capable of inhibition of cellular secretion, passes
into the cytosol of the target cell by cellular internalization
mechanisms. Thereafter, inhibition of secretion from the target cell is
effected.
AN 2001:228744 HCAPLUS <<LOGINID::20071210>>
DN 134:247267
TI Clostridial neurotoxin targeted conjugates for inhibition of secretion
from non-neuronal cells
IN Foster, Keith Alan; Chaddock, John Andrew; Purkiss, John Robert; Quinn,
Conrad Padraig
PA Microbiological Research Authority, UK

SO PCT Int. Appl., 63 pp.
 CODEN: PIXXD2
 DT Patent
 LA English
 FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001021213	A2	20010329	WO 2000-GB3669	20000925 <--
	WO 2001021213	A3	20020711		
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	CA 2383470	A1	20010329	CA 2000-2383470	20000925 <--
	EP 1235594	A2	20020904	EP 2000-962721	20000925 <--
	R:			AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL	
	JP 2003509476	T	20030311	JP 2001-524636	20000925 <--
	AU 782457	B2	20050728	AU 2000-74365	20000925 <--
	US 2003180289	A1	20030925	US 2002-88665	20020814 <--
	AU 2005227383	A1	20051124	AU 2005-227383	20051027 <--
	US 2006216283	A1	20060928	US 2006-327855	20060109 <--
PRAI	GB 1999-22554	A	19990923	<--	
	WO 2000-GB3669	W	20000925	<--	
	WO 2000-GB3681	A	20000925	<--	
	US 2002-88665	A1	20020814		

L30 ANSWER 2 OF 3 HCAPLUS - COPYRIGHT 2007 ACS on STN

TI Interactions of *Penicillium marneffei* with human leukocytes in vitro
 AB *Penicillium marneffei*, a dimorphic fungus endemic in parts of Asia, causes disease in those with impaired cell-mediated immunity, especially persons with AIDS. The histopathol. of penicilliosis *marneffei* features the intracellular infection of macrophages. The authors studied the interactions between human leukocytes and heat-killed yeast-phase *P. marneffei*. Monocyte-derived macrophages bound and internalized *P. marneffei* in the presence of complement-sufficient pooled human serum (PHS). Binding and phagocytosis were still seen if PHS was heat inactivated or omitted altogether. The binding of unopsonized *P. marneffei* to monocyte-derived macrophages occurred in the absence of divalent cations and was not affected by inhibitors of mannose and . beta.-glucan receptors or monoclonal antibodies directed against CD14 and CD11/CD18. Binding was profoundly inhibited by wheat germ agglutinin. A vigorous respiratory burst was seen in peripheral blood mononuclear cells (PBMC) stimulated with *P. marneffei*, regardless of whether the fungi were opsonized. However, tumor necrosis factor alpha (TNF- α) release from PBMC stimulated with *P. marneffei* occurred only if serum was present. These data demonstrate that (i) monocyte-derived macrophages bind and phagocytose *P. marneffei* even in the absence of opsonization, (ii) binding is divalent cation independent but is inhibited by wheat germ agglutinin, suggesting that the major receptor(s) recognizing *P. marneffei* is a glycoprotein with exposed N-acetyl- β -D-glucosaminyl groups, (iii) *P. marneffei* stimulates the respiratory burst regardless of whether opsonins are present, and (iv) serum factors are required for *P. marneffei* to stimulate TNF- α release. The ability of unopsonized *P. marneffei* to parasitize mononuclear phagocytes without stimulating the production of TNF- α may be critical for the virulence of this intracellular parasite.

AN 1999:554591 HCAPLUS <<LOGINID::20071210>>
 DN 131:285214

TI Interactions of *Penicillium marneffei* with human leukocytes in vitro
AU Rongrungruang, Yong; Levitz, Stuart M.
CS The Evans Memorial Department of Clinical Research and the Department of
Medicine, Boston University School of Medicine, Boston, MA, 02118, USA
SO Infection and Immunity (1999), 67(9), 4732-4736
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English

RE.CNT 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 3 OF 3 HCAPLUS COPYRIGHT 2007 ACS on STN

TI The β -glucan-binding lectin site of mouse CR3
(CD11b/CD18) and its function in generating a primed state of the receptor
that mediates cytotoxic activation in response to iC3b-opsonized target
cells
AB Mouse leukocyte CR3 (Mac-1, α M β 2 integrin) was shown to
function as a receptor for β -glucans in the same way as human CR3.
Soluble zymosan polysaccharide (SZP) or pure β -glucans labeled with FITC
or 125I bound in a saturable and reversible manner to neutrophils,
macrophages, and NK cells. This lectin activity was blocked by anti-CD11b
mAb M1/70 or 5C6 and did not occur with leukocytes from CR3-/-
(CD11b-deficient) mice. SZP preps. containing primarily mannose or glucose
bound to CR3, and the binding of 125I-labeled β -
glucan to CR3 was competitively inhibited by β -glucans from
barley or seaweed, but not by yeast α -mannan. Also, as with
human CR3, the lectin site of mouse CR3 was inhibited by α - or
 β -methylglucoside (but not D-glucose), α - or
 β -methylmannoside, and N-acetyl-D-glucosamine. Phagocytosis of
zymosan and serum-opsonized zymosan was partially inhibited by anti-CR3
and was reduced to <40% of normal with leukocytes from CR3-/- mice. As
with neutrophils from patients with CD18 deficiency, neutrophils from
CR3-/- mice exhibited no phagocytosis of particulate β -
glucan. SZP or β -glucans primed CR3 of neutrophils,
macrophages, and NK cells for cytotoxicity of iC3b-opsonized tumor
cells that otherwise did not trigger killing. β -
Glucan priming for cytotoxicity was inhibited by anti-CR3 and did
not occur with leukocytes from CR3-/- mice. The primed state of
macrophage and NK cell CR3 remained detectable for 18 to 24 h after
pulsing with β -glucans. The similarity of mouse and human CR3 in
response to β -glucans highlights the utility of mouse tumor
models for development of therapeutic β -glucans.

AN 1999:107663 HCAPLUS <<LOGINID::20071210>>

DN 130:280682

TI The β -glucan-binding lectin site of mouse CR3
(CD11b/CD18) and its function in generating a primed state of the receptor
that mediates cytotoxic activation in response to iC3b-opsonized target
cells

AU Xia, Yu; Vetvicka, Viclav; Yan, Jun; Hanikyrova, Margareta; Mayadas,
Tanya; Ross, Gordon D.

CS Division of Experimental Immunology and Immunopathology, Department of
Pathology, and Department of Microbiology and Immunology, University of
Louisville, Louisville, KY, 40292, USA

SO Journal of Immunology (1999), 162(4), 2281-2290
CODEN: JOIMA3; ISSN: 0022-1767

PB American Association of Immunologists

DT Journal

LA English

RE.CNT 83 THERE ARE 83 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d l32 1-2 ti abs bib

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L32 ANSWER 1 OF 2 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Clostridial neurotoxin targeted conjugates for inhibition of secretion from non-neuronal cells

AB A method of treatment of disease by inhibition of cellular secretory processes is provided. The method has particular application in the treatment of diseases dependent on the exocytotic activity of endocrine cells, exocrine cells, inflammatory cells, cells of the immune system, cells of the cardiovascular system, and bone cells. Agents and compns. therefor, as well as methods for manufacturing these agents and compns., are provided. In a preferred embodiment a clostridial neurotoxin, substantially devoid of holotoxin binding affinity for neuronal cells of the presynaptic muscular junction, is associated with a targeting moiety. The targeting moiety is selected such that the clostridial toxin conjugate so formed may be directed to a non-neuronal target cell to which the conjugate may bind. Following binding, a neurotoxin component of the conjugate, which is capable of inhibition of cellular secretion, passes into the cytosol of the target cell by cellular internalization mechanisms. Thereafter, inhibition of secretion from the target cell is effected.

AN 2001:228744 HCAPLUS <<LOGINID::20071210>>

DN 134:247267

TI Clostridial neurotoxin targeted conjugates for inhibition of secretion from non-neuronal cells

IN Foster, Keith Alan; Chaddock, John Andrew; Purkiss, John Robert; Quinn, Conrad Padraig

PA Microbiological Research Authority, UK

SO PCT Int. Appl., 63 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001021213	A2	20010329	WO 2000-GB3669	20000925 <--
	WO 2001021213	A3	20020711		
	W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW			
	RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
	CA 2383470	A1	20010329	CA 2000-2383470	20000925 <--
	EP 1235594	A2	20020904	EP 2000-962721	20000925 <--
	R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL			
	JP 2003509476	T	20030311	JP 2001-524636	20000925 <--
	AU 782457	B2	20050728	AU 2000-74365	20000925 <--
	US 2003180289	A1	20030925	US 2002-88665	20020814 <--
	AU 2005227383	A1	20051124	AU 2005-227383	20051027 <--
	US 2006216283	A1	20060928	US 2006-327855	20060109 <--
PRAI	GB 1999-22554	A	19990923	<--	
	WO 2000-GB3669	W	20000925	<--	
	WO 2000-GB3681	A	20000925	<--	
	US 2002-88665	A1	20020814		

L32 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Interactions of Penicillium marneffei with human leukocytes in vitro

AB *Penicillium marneffei*, a dimorphic fungus endemic in parts of Asia, causes disease in those with impaired cell-mediated immunity, especially persons with AIDS. The histopathol. of penicilliosis *marneffei* features the intracellular infection of macrophages. The authors studied the interactions between human leukocytes and heat-killed yeast-phase *P. marneffei*. Monocyte-derived macrophages bound and internalized *P. marneffei* in the presence of complement-sufficient pooled human serum (PHS). Binding and phagocytosis were still seen if PHS was heat inactivated or omitted altogether. The binding of unopsonized *P. marneffei* to monocyte-derived macrophages occurred in the absence of divalent cations and was not affected by inhibitors of mannose and β -glucan receptors or monoclonal antibodies directed against CD14 and CD11/CD18. Binding was profoundly inhibited by wheat germ agglutinin. A vigorous respiratory burst was seen in peripheral blood mononuclear cells (PBMC) stimulated with *P. marneffei*, regardless of whether the fungi were opsonized. However, tumor necrosis factor alpha (TNF- α) release from PBMC stimulated with *P. marneffei* occurred only if serum was present. These data demonstrate that (i) monocyte-derived macrophages bind and phagocytose *P. marneffei* even in the absence of opsonization, (ii) binding is divalent cation independent but is inhibited by wheat germ agglutinin, suggesting that the major receptor(s) recognizing *P. marneffei* is a glycoprotein with exposed N-acetyl- β -D-glucosaminyl groups, (iii) *P. marneffei* stimulates the respiratory burst regardless of whether opsonins are present, and (iv) serum factors are required for *P. marneffei* to stimulate TNF- α release. The ability of unopsonized *P. marneffei* to parasitize mononuclear phagocytes without stimulating the production of TNF- α may be critical for the virulence of this intracellular parasite.

AN 1999:554591 HCAPLUS <<LOGINID::20071210>>

DN 131:285214

TI Interactions of *Penicillium marneffei* with human leukocytes in vitro

AU Rongrungruang, Yong; Levitz, Stuart M.

CS The Evans Memorial Department of Clinical Research and the Department of Medicine, Boston University School of Medicine, Boston, MA, 02118, USA

SO Infection and Immunity (1999), 67(9), 4732-4736

CODEN: INFIBR; ISSN: 0019-9567

PB American Society for Microbiology

DT Journal

LA English

RE.CNT 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d 129 1-7 ti

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L29 ANSWER 1 OF 7 HCAPLUS COPYRIGHT 2007 ACS on STN

TI *Coniothyrium minitans* β -(1,3) exoglucanase gene *cbeG1*

L29 ANSWER 2 OF 7 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Clostridial neurotoxin targeted conjugates for inhibition of secretion from non-neuronal cells

L29 ANSWER 3 OF 7 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Interactions of *Penicillium marneffei* with human leukocytes in vitro

L29 ANSWER 4 OF 7 HCAPLUS COPYRIGHT 2007 ACS on STN

TI The β -glucan-binding lectin site of mouse CR3 (CD11b/CD18) and its function in generating a primed state of the receptor that mediates cytotoxic activation in response to iC3b-opsonized target cells

L29 ANSWER 5 OF 7 HCAPLUS COPYRIGHT 2007 ACS on STN
 TI Analysis of the sugar specificity and molecular location of the .
 beta.-glucan-binding lectin site of complement
 receptor type 3 (CD11b/CD18)

L29 ANSWER 6 OF 7 HCAPLUS COPYRIGHT 2007 ACS on STN
 TI Specificity of membrane complement receptor type three (CR3) for
 β-glucans

L29 ANSWER 7 OF 7 HCAPLUS COPYRIGHT 2007 ACS on STN
 TI Activation of human polymorphonuclear leukocytes by particulate zymosan is
 related to both its major carbohydrate components: glucan and mannan

=> d l5-l7 ti abs bib
 'L5-L7' IS NOT A VALID ACCESSION NUMBER
 'TI' IS NOT A VALID ACCESSION NUMBER
 'ABS' IS NOT A VALID ACCESSION NUMBER
 'BIB' IS NOT A VALID ACCESSION NUMBER

The number entered is not a valid accession number in this file.
 Enter "HELP ACCESSION" at an arrow prompt (=>) for a list of valid
 accession number formats in the current file.

=> y
 Y IS NOT A RECOGNIZED COMMAND
 The previous command name entered was not recognized by the system.
 For a list of commands available to you in the current file, enter
 "HELP COMMANDS" at an arrow prompt (=>).

=> d l29 5-7 ti abs bib
 YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L29 ANSWER 5 OF 7 HCAPLUS COPYRIGHT 2007 ACS on STN
 TI Analysis of the sugar specificity and molecular location of the .
 beta.-glucan-binding lectin site of complement
 receptor type 3 (CD11b/CD18)

AB Zymosan, the cell wall from *Saccharomyces cerevisiae*, was reported to be a
 macrophage activator through its β-glycan over 30 yr ago.
 Nevertheless, the identity of the β -glucan
 receptor has been controversial. This study showed that the
 αβ2-integrin, CR3 (Mac-1, CD11b/CD18) served as the .
 beta.-glucan receptor through one or more lectin sites
 located outside of the CD11b I-domain that contains the binding sites for
 iC3b, ICAM-1, and fibrinogen. Sugar specificity, analyzed with
 FITC-labeled soluble polysaccharides and flow cytometry, showed CR3-specific
 staining with several pure β-glucans but not with α-mannan.
 However, a 10-kDa soluble zymosan polysaccharide (SZP) with high affinity
 (6.7+10-8M) for CR3 consisted largely of mannose and .apprx.5%
 glucose. Binding of either SZP-FITC or β -glucan
 -FITC to CR3 was blocked not only by pure β-glucans from yeast,
 mushroom, seaweed, or barley, but also by N-acetyl-D-glucosamine
 (NADG), α- or β-methylmannoside, and α- or
 β-methylglucoside. SZP-FITC and β -glucan
 -FITC stained all leukocyte types similarly to anti-CR3-FITC, and
 polysaccharide-FITC staining was inhibited ≥95% by unlabeled
 anti-CR3. SZP-FITC staining of cells expressing recombinant chimeras
 between CR3 and CR4 (p150,95, CD11c/CD18) suggested that both the divalent
 cation-binding region of CD11b and the region C-terminal to it may
 regulate binding of polysaccharides to CR3. Unlabeled SZP or .
 beta.-glucan also blocked CR3 staining by 11 mAb to
 C-terminal domain epitopes of CD11b but had no effect on staining by mAb

directed to the 1-domain. In conclusion, CR3 serves as the leukocyte .
beta.-glucan receptor through a cation-independent
lectin site located C-terminal to the 1-domain of CD11b. Its sugar
specificity is broader than originally appreciated, allowing it to react
with certain polysaccharides containing mannose or NADG, as well as glucose.

AN 1996:63811 HCAPLUS <<LOGINID::20071210>>

DN 124:114996

TI Analysis of the sugar specificity and molecular location of the .
beta.-glucan-binding lectin site of complement
receptor type 3 (CD11b/CD18)

AU Thornton, Brian P.; Vetvicka, Vaclav; Pitman, Mark; Goldman, Robert C.;
Ross, Gordon D.

CS Dep. Pathol., Univ. Louisville, Louisville, KY, 40292, USA

SO Journal of Immunology (1996), 156(3), 1235-46

CODEN: JOIMA3; ISSN: 0022-1767

PB American Association of Immunologists

DT Journal

LA English

L29 ANSWER 6 OF 7 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Specificity of membrane complement receptor type three (CR3) for
β-glucans

AB The binding of the iC3b receptor (CR3) to unopsonized zymosan resulted
from CR3 attachment to cell wall β-glucans. A specificity of
neutrophil responses for β -glucan was first
suggested by a comparison of yeast (*Saccharomyces cerevisiae*) cell wall
components for stimulation of a neutrophil superoxide burst. Three types
of expts. demonstrated a role for CR3 in these responses. First,
neutrophil ingestion of either yeast or yeast-derived β -
glucan particles was blocked by monoclonal anti-CR3, fluid-phase
iC3b, or soluble β -glucan from barley.
Monocyte ingestion of β -glucan particles was also
blocked by anti-CR3, but not by anti-CR1 or anti-C3. Second, the
neutrophil superoxide burst response to either zymosan or .beta
.-glucan particles was blocked by anti-CR3 or fluid-phase iC3b,
and was completely absent with neutrophils from 3 patients with an
inherited deficiency of CR3. Third, CR3 was isolated from solubilized
neutrophils by affinity chromatog. on β -glucan
-Sephrose.

AN 1987:552442 HCAPLUS <<LOGINID::20071210>>

DN 107:152442

TI Specificity of membrane complement receptor type three (CR3) for
β-glucans

AU Ross, Gordon D.; Cain, Judith A.; Myones, Barry L.; Newman, Simon L.;
Lachmann, Peter J.

CS Dep. Med., Univ. North Carolina, Chapel Hill, NC, 27514, USA

SO Complement (1987), 4(2), 61-74

CODEN: CMPLDF; ISSN: 0253-5076

DT Journal

LA English

L29 ANSWER 7 OF 7 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Activation of human polymorphonuclear leukocytes by particulate zymosan is
related to both its major carbohydrate components: glucan and mannan

AB Unopsonized particulate zymosan and its major carbohydrate component
glucan were phagocytosed under serum-free conditions by adherent
polymorphonuclear leukocytes (PMN) in a dose- and time-dependent manner.
Preincubation of PMN monolayers with mannan did not cause a reduction in the
phagocytosis of either particle. The phagocytic response was inhibited by
preincubation of the cells with trypsin at a concentration that did not inhibit
the phagocytosis of sheep erythrocytes coated with IgG or of latex
particles. Homol. of the recognition mechanisms for glucan and zymosan
was confirmed when cells cultured on fixed glucan or on fixed zymosan
failed to ingest either particle to more than 40% of control phagocytosis.

Similarly, zymosan and glucan activated PMN in suspension, in a dose- and time-dependent manner, to generate reactive O species which were measured as luminol-dependent chemiluminescence (CL). There was however, a 4-fold greater CL response to zymosan. Preincubation of PMN with mannan resulted in a decreased CL response to zymosan, while the response to glucan was unaffected. The CL response was also sensitive to a range of concns. of trypsin. In contrast, 2 other complex polysaccharide particles (barley-derived β -glucan and algae-derived laminarin) were not phagocytosed by PMN, nor did they cause the generation of CL, despite the fact that they possessed the capacity, in common with zymosan and glucan, to activate the alternative pathway of complement. The identification of a trypsin-sensitive recognition mechanism on the surface of human PMN for unopsonized zymosan and glucan represents a response not hitherto characterized. Furthermore, the phagocytosis of unopsonized zymosan by human PMN is dependent primarily on its glucan content, but its capacity to activate the respiratory burst may involve mannan and the recruitment of a second cell surface recognition mechanism.

AN 1986:404970 HCAPLUS <<LOGINID::20071210>>
 DN 105:4970
 TI Activation of human polymorphonuclear leukocytes by particulate zymosan is related to both its major carbohydrate components: glucan and mannan
 AU Williams, J. D.; Topley, N.; Alobaidi, H. M.; Harber, M. J.
 CS KRUF Inst., R. Infirm., Cardiff, UK
 SO Immunology (1986), 58(1), 117-24
 CODEN: IMMUAM; ISSN: 0019-2805
 DT Journal
 LA English

=> d his

(FILE 'HOME' ENTERED AT 16:02:28 ON 10 DEC 2007)

FILE 'HCAPLUS' ENTERED AT 16:11:01 ON 10 DEC 2007

L1 4854 S BETA-GLUCAN
 L2 71149 S COMPLEMENT
 L3 363997 S ANTIBODY OR MONOCLONAL
 L4 52754 S BARLEY
 L5 146 S L1 AND L2
 L6 9 S L1 AND L2 AND L4
 L7 210 S L1 AND L3
 L8 17 S L1 AND L3 AND L4
 L9 48 S L1 AND L2 AND L3
 L10 4 S L1 AND L2 AND L3 AND L4

FILE 'STNGUIDE' ENTERED AT 16:11:12 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 16:11:58 ON 10 DEC 2007

L11 5 S L6 AND (PY<2002 OR AY<2002 OR PRY<2002)
 L12 9 S L8 AND (PY<2002 OR AY<2002 OR PRY<2002)
 L13 17 S L9 AND (PY<2002 OR AY<2002 OR PRY<2002)
 L14 1 S L10 AND (PY<2002 OR AY<2002 OR PRY<2002)

FILE 'STNGUIDE' ENTERED AT 16:12:11 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 16:12:19 ON 10 DEC 2007

FILE 'STNGUIDE' ENTERED AT 16:12:20 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 16:12:51 ON 10 DEC 2007

FILE 'STNGUIDE' ENTERED AT 16:12:52 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 16:13:10 ON 10 DEC 2007

FILE 'STNGUIDE' ENTERED AT 16:13:10 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 16:13:26 ON 10 DEC 2007

FILE 'STNGUIDE' ENTERED AT 16:13:26 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 16:16:02 ON 10 DEC 2007

L15 795839 S (CANCER OR TUMOR OR NEOPLAS?)
L16 57 S L15 AND L5
L17 1 S L15 AND L11
L18 57 S L15 AND L7
L19 0 S L15 AND L12
L20 6 S L15 AND L13
L21 0 S L15 AND L14
L22 29 S L16 AND (PY<2002 OR AY<2002 OR PRY<2002)
L23 1 S L17 AND (PY<2002 OR AY<2002 OR PRY<2002)

FILE 'STNGUIDE' ENTERED AT 16:16:17 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 16:18:56 ON 10 DEC 2007

FILE 'STNGUIDE' ENTERED AT 16:18:56 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 16:19:03 ON 10 DEC 2007

FILE 'STNGUIDE' ENTERED AT 16:19:04 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 16:19:15 ON 10 DEC 2007

FILE 'STNGUIDE' ENTERED AT 16:19:16 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 17:08:53 ON 10 DEC 2007

L24 191167 S BARLEY OR OAT OR WHEAT OR MOSS
L25 11 S L24 AND L1 AND L2
L26 5 S L24 AND L1 AND L2 AND L15
L27 25 S L24 AND L1 AND L3
L28 5 S L24 AND L1 AND L3 AND L15
L29 7 S L25 AND (PY<2002 OR AY<2002 OR PRY<2002)
L30 3 S L26 AND (PY<2002 OR AY<2002 OR PRY<2002)
L31 14 S L27 AND (PY<2002 OR AY<2002 OR PRY<2002)
L32 2 S L28 AND (PY<2002 OR AY<2002 OR PRY<2002)

FILE 'STNGUIDE' ENTERED AT 17:09:11 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 17:09:22 ON 10 DEC 2007

FILE 'STNGUIDE' ENTERED AT 17:09:22 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 17:09:42 ON 10 DEC 2007

FILE 'STNGUIDE' ENTERED AT 17:09:42 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 17:10:10 ON 10 DEC 2007

FILE 'STNGUIDE' ENTERED AT 17:10:10 ON 10 DEC 2007

FILE 'HCAPLUS' ENTERED AT 17:11:03 ON 10 DEC 2007

FILE 'STNGUIDE' ENTERED AT 17:11:03 ON 10 DEC 2007

=> log hold

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

FULL ESTIMATED COST	ENTRY 0.06	SESSION 110.35
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-16.38

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STN INTERNATIONAL SESSION SUSPENDED AT 17:11:10 ON 10 DEC 2007

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Welcome to STN International! Enter x:x

LOGINID:SSPTAEXO1623

PASSWORD:

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FILE 'STNGUIDE' ENTERED AT 17:34:41 ON 10 DEC 2007
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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	110.35
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-16.38

=> file hcaplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	110.35
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-16.38

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FILE LAST UPDATED: 7 Dec 2007 (20071207/ED)

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substance identification.

=> s cr3

L33 19379 CR3

=> s l13 and l33

L34 9 L13 AND L33

=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	2.60	112.95
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
	ENTRY	SESSION
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=> d l34 1-9 ti

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L34 ANSWER 1 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Clostridial neurotoxin targeted conjugates for inhibition of secretion
from non-neuronal cells

L34 ANSWER 2 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Nonopsonic phagocytosis of zymosan and Mycobacterium kansasii by
CR3 (CD11b/CD18) involves distinct molecular determinants and is
or is not coupled with NADPH oxidase activation

L34 ANSWER 3 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Inhibition of interleukin-12 production using ligands for CD46 or
complement receptor CR3

L34 ANSWER 4 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Contribution of CR3 to nitric oxide production from macrophages
stimulated with high-dose of LPS

L34 ANSWER 5 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Activation, binding, and processing of complement component 3
(C3) by Blastomyces dermatitidis

L34 ANSWER 6 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI The function of human NK cells is enhanced by β -
glucan, a ligand of CR3 (CD11b/CD18)

L34 ANSWER 7 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Role of complement receptor type three and serum opsonins in the
neutrophil response to yeast

L34 ANSWER 8 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Specificity of membrane complement receptor type three (
CR3) for β -glucans

L34 ANSWER 9 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Role of the adherence-promoting receptors, CR3, LFA-1, and
p150,95, in binding of Histoplasma capsulatum by human macrophages

=> d l34 d l34 4 6 7 8 9 ti abs bib
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=> d l34 4 6 7 8 9 ti abs bib
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L34 ANSWER 4 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Contribution of CR3 to nitric oxide production from macrophages
stimulated with high-dose of LPS

AB The contribution of the complement receptor type 3 (CR3
) to nitric oxide (NO) production from macrophages stimulated by LPS was
investigated. When thioglycollate-elicited mouse peritoneal macrophages
were stimulated with a high dose of LPS (10 µg/mL) in both the presence
and absence of fetal calf serum, a source of LPS binding protein (LBP)
necessary for the binding of LPS to CD14, NO production was observed. These
findings suggest that CD14-dependent and CD14-independent signaling
pathways for NO production are present in macrophages. Because binding and
phagocytosis of bacteria by macrophages through the CR3 has been
previously reported, we investigated whether the CR3 acts in
CD14-independent signaling pathway for NO production. By flow cytometric
anal., the binding of FITC-labeled anti-CR3 monoclonal
antibody (anti-CR3 mAb) to macrophages was inhibited by
LPS. Anti-CR3 mAb induced iNOS protein and produced NO in a
dose dependent manner. Further, NO production induced by anti-CR3
mAb was also inhibited by zymocel, β-glucan with
a high affinity to CR3. These results suggest that the
CR3 mol. acts in a CD14-independent signaling pathway, and
contributes to NO production by macrophages stimulated with high doses of LPS.

AN 1998:174595 HCAPLUS <<LOGINID::20071210>>
DN 128:307329

TI Contribution of CR3 to nitric oxide production from macrophages
stimulated with high-dose of LPS

AU Matsuno, Ryozi; Aramaki, Yukihiro; Arima, Hidetoshi; Adachi, Yoshiyuki;
Ohno, Naohito; Yadomae, Toshiro; Tsuchiya, Seishi

CS School of Pharmacy, Tokyo University of Pharmacy and Life Science, Tokyo,
192-03, Japan

SO Biochemical and Biophysical Research Communications (1998),
244(1), 115-119
CODEN: BBRCA9; ISSN: 0006-291X

PB Academic Press

DT Journal

LA English

RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L34 ANSWER 6 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN

TI The function of human NK cells is enhanced by β -
glucan, a ligand of CR3 (CD11b/CD18)

AB Cells responsible for the natural killer (NK) effect in human blood can be

collected in the low-d. lymphocyte subset and the majority of them express the CR3 complement receptor. In addition to the iC3b binding site, the CR3 mols. possess an epitope which binds . beta.-glucan. Ligands of this site can deliver activation signals to CR3-carrying monocytes and neutrophils. The function of NK cells was also potentiated by preincubation with . beta.-glucan. The treatment increased the proportion of target-binding lymphocytes and of the damaged target cells in the conjugates. The monoclonal antibody OKM-1, directed to the β -glucan-binding site of CR3, abrogated this effect. Another CR3-reactive monoclonal antibody, M522, known to activate monocytes and neutrophils, enhanced the NK function.

AN 1991:605839 HCAPLUS <<LOGINID::20071210>>

DN 115:205839

TI The function of human NK cells is enhanced by β - glucan, a ligand of CR3 (CD11b/CD18)

AU Di Renzo, Livia; Yefenof, Eitan; Klein, Eva

CS Dep. Tumor Biol., Karolinska Inst., Stockholm, S-104 01, Swed.

SO European Journal of Immunology (1991), 21(7), 1755-8

CODEN: EJIMAF; ISSN: 0014-2980

DT Journal

LA English

L34 ANSWER 7 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Role of complement receptor type three and serum opsonins in the neutrophil response to yeast

AB Neutrophil complement receptor type 3 (CR3) was shown to play a major role in the phagocytic and respiratory burst response to serum-opsonized yeast (OY). The neutrophil response to OY was greatly reduced by monoclonal anti-CR3, and when Y was opsonized with purified complement components instead of serum, YC3b(i) stimulated neutrophil responses of a similar magnitude to OY. The mechanism of the neutrophil response to OY apparently involves 3 stages: (1) high-avidity binding of particles via fixed iC3b and iC3b binding site of CR3, (2) low-avidity binding of glucan sugars in the Y cell wall to the β -glucan binding site of CR3, and (3) stimulation of ingestion and a respiratory burst via the . beta.-glucan binding site of CR3. Only minor contributions of CR1 and Fc receptors could be demonstrated, despite the presence of fixed C3b and IgG on the OY.

AN 1987:573918 HCAPLUS <<LOGINID::20071210>>

DN 107:173918

TI Role of complement receptor type three and serum opsonins in the neutrophil response to yeast

AU Cain, Judith A.; Newman, Simon L.; Ross, Gordon D.

CS Dep. Med., Univ. North Carolina, Chapel Hill, NC, 27514, USA

SO Complement (1987), 4(2), 75-86

CODEN: CMLPDL; ISSN: 0253-5076

DT Journal

LA English

L34 ANSWER 8 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Specificity of membrane complement receptor type three (CR3) for β -glucans

AB The binding of the iC3b receptor (CR3) to unopsonized zymosan resulted from CR3 attachment to cell wall β -glucans. A specificity of neutrophil responses for β -glucan was first suggested by a comparison of yeast (*Saccharomyces cerevisiae*) cell wall components for stimulation of a neutrophil superoxide burst. Three types of expts. demonstrated a role for CR3 in these responses. First, neutrophil ingestion of either yeast or yeast-derived . beta.-glucan particles was blocked by monoclonal anti-CR3, fluid-phase iC3b, or soluble β -

glucan from barley. Monocyte ingestion of β - glucan particles was also blocked by anti-CR3, but not by anti-CR1 or anti-C3. Second, the neutrophil superoxide burst response to either zymosan or β -glucan particles was blocked by anti-CR3 or fluid-phase iC3b, and was completely absent with neutrophils from 3 patients with an inherited deficiency of CR3. Third, CR3 was isolated from solubilized neutrophils by affinity chromatog. on β -glucan -Sephrose.

AN 1987:552442 HCAPLUS <<LOGINID::20071210>>

DN 107:152442

TI Specificity of membrane complement receptor type three (CR3) for β -glucans

AU Ross, Gordon D.; Cain, Judith A.; Myones, Barry L.; Newman, Simon L.; Lachmann, Peter J.

CS Dep. Med., Univ. North Carolina, Chapel Hill, NC, 27514, USA

SO Complement (1987), 4(2), 61-74

CODEN: CMPLDF; ISSN: 0253-5076

DT Journal

LA English

L34 ANSWER 9 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Role of the adherence-promoting receptors, CR3, LFA-1, and p150,95, in binding of Histoplasma capsulatum by human macrophages

AB The principal host cell of H. capsulatum (Hc) is the macrophage (M.vphi.) within which the pathogenic yeast phase of the fungus multiplies during active disease. The major receptor mechanism that mediates the attachment of unopsonized Hc yeasts to human monocyte-derived M.vphi. from peripheral blood was identified. Binding of Hc yeasts by M.vphi. is rapid, temperature-dependent, and requires both Ca and Mg ions for optimum activity. Recognition of Hc yeasts does not require Fc receptors, mannosyl/fucosyl receptors, β -glucan receptors, or secretion of complement C3 by M.vphi. Studies were performed on the effect of down-regulating specific receptors of the CR3/LFA-1/p150,95 adherence-promoting protein family from the apical portion of M.vphi. to determine the effects upon binding of Hc yeasts. Anti- β chain monoclonal antibodies that recognize all 3 of these proteins blocked binding of yeasts. However, removal of individual receptors with antibodies against the α polypeptides caused negligible depression of binding, and removal of any pair caused only modest depression. Thus, each of the members of the CR3/LFA-1/p150,95 family is independently capable of binding Hc.

AN 1987:82904 HCAPLUS <<LOGINID::20071210>>

DN 106:82904

TI Role of the adherence-promoting receptors, CR3, LFA-1, and p150,95, in binding of Histoplasma capsulatum by human macrophages

AU Bullock, Ward E.; Wright, Samuel D.

CS Lab. Cell. Physiol. Immunol., Rockefeller Univ., New York, NY, 10021, USA

SO Journal of Experimental Medicine (1987), 165(1), 195-210

CODEN: JEMEAV; ISSN: 0022-1007

DT Journal

LA English